Rupture of abdominal aortic aneurysm: Concurrent comparison of outcome of those occurring after endovascular repair versus those occurring without previous treatment in an 11-year single-center experience

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Objective: **The purpose of this single-center study was to compare findings at presentation and surgical outcome in patients in whom abdominal aortic aneurysms (AAAs) ruptured after endovascular repair and patients in whom AAAs ruptured before any treatment, over a defined period.**

Methods: **From May 1992 to September 2003, 1043 patients underwent elective repair of intact infrarenal AAAs. Endovascular repair was performed in 609 patients, and open repair in 434 patients. Eighteen of 609 patients (3%) who underwent endovascular AAA repair required treatment because of rupture of the aneurysm after a mean of 29 months (group 1). During the same 11-year period, another 91 patients without previous treatment required urgent repair of a ruptured AAA (group 2). Rupture was diagnosed at contrast material–enhanced computed tomography or by presence of extramural extravasation of blood at open repair. Except for a higher incidence of women in group 2, patients in both groups were similar with regard to demographics and clinical characteristics but differed in findings at presentation. Eight patients in group 1 had a known endoleak before AAA rupture, whereas contrast-enhanced computed tomography, performed in 15 patients at presentation, demonstrated an endoleak in all. Hypotension (systolic blood pressure <100 mm Hg) was noted at presentation in 4 of 18 patients (22%) in group 1 and 76 of 91 patients (84%) in group 2. All patients underwent open repair via a transperitoneal approach, except for 4 patients in group 1 and 3 patients in group 2 who underwent endovascular repair of ruptured AAAs.**

Results: **The proportion of patients with hypotension at presentation in group 1 (4 of 18) was significantly less than in group 2 (76 of 91; $P < .01$). The difference in perioperative (30 day) mortality rate in group 1 (3 of 18; 16.6%) compared with group 2 (49 of 91; 53.8%) was also significant ($P < .01$). The outcome in group 1 was therefore superior to that in group 2.**

Conclusions: **This study confirms that endovascular AAA repair complicated by endoleak does not prevent rupture. The data suggest, however, that rupture, when it occurs in these circumstances, may not be accompanied by such major hemodynamic changes and high mortality as rupture of an untreated AAA. Further long-term follow-up and analysis in a larger group of patients are required to confirm the apparent intermediate level of protection afforded by failed endovascular repair, which does not prevent rupture but enhances survival after operation to treat rupture, possibly by ameliorating the hemodynamic changes associated with the rupture process. (J Vasc Surg 2004;40:860-6.)**

We have previously reported unexpected survival in 4 patients at high risk who underwent open repair of late rupture of an abdominal aortic aneurysm (AAA) after previous endovascular repair.[1]

All 4 patients had a known endoleak, and the aneurysm ruptured at a mean of 10 months after endovascular repair. We have subsequently observed rupture after AAA repair in patients with no previous endoleak. Our continuing experience of noting survival in patients at high risk in whom this outcome would not have been anticipated led to this subsequent concurrent comparison of outcome of ruptured AAA occurring after endovascular repair versus those occurring de novo, over 11 years.

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OBJECTIVE

The purpose of this single-center study was to compare findings at presentation and surgical outcome in patients in whom AAAs ruptured after endovascular repair versus those in whom AAAs ruptured before any treatment, over a defined period of time.

METHODS

Between May 1992 and September 2003, 1043 patients underwent elective repair of intact infrarenal AAAs at

Royal Prince Alfred Hospital. Endovascular repair was performed in 609 patients, and open repair was performed in 434 patients. Eighteen of 609 patients (3%) who underwent endovascular AAA repair required treatment because of rupture of the aneurysm at a mean of 29 months (range, 4 days–6.5 years) after surgery (group 1; Tables I and II). Over the same 11 years another 91 patients without previous treatment required urgent repair of ruptured AAAs (group 2). Rupture was diagnosed at contrast material–enhanced computed tomography (CT) or by the presence of extramural extravasation of blood observed at open repair. Except for a higher proportion of women in group 2, demographic and clinical characteristics were similar in both groups (Table III), but findings at presentation differed. In group 1, 8 patients had known endoleaks before rupture. These were type I endoleaks, 7 proximal and 1 distal, associated with the iliac limb of a bifurcated graft. Three patients had endotension with expanding aneurysms, but no evidence of endoleak. Five patients attended regular follow-up, and had no endoleak and no expansion of the aneurysm. The remaining 2 patients failed to return for follow-up despite explanation of its importance and being given reminders.

Ten patients in group 1 had co-morbid conditions that were considered to put them at high risk and unfit for open operation at the primary AAA repair (Table IV). Similar assessment was difficult in patients in group 2, because initial presentation was acute, with a life-threatening condition.

Presentation. The intensity of abdominal and back pain at presentation was initially similar in both groups, but patients in group 1 experienced less pain after this, and were able to move with greater freedom compared with patients in group 2. Hypotension, defined as systolic blood pressure less than 100 mm Hg, was noted at presentation in 4 of 18 patients (22%) in group 1 and 76 of 91 patients (84%) in group 2. Median duration from presentation to intervention in group 1 was 8 hours (range, 2-140 hours) and in group 2 was 4.5 hours (range, 1-23 hours).

CT. Triple-phase studies were performed at 48 hours and at 6 and 12 months after operation, and annually thereafter.

Sixteen patients in group 1 underwent CT at presentation; mean AAA diameter at this time was 6.9 cm. Sac size had increased in all but 2 patients, compared with the diameter before endovascular AAA repair.

Endoleak was noted in 15 of 16 patients in group 1 in whom CT scans were obtained at presentation, including the 3 patients with endotension. Patient 16, with chronic renal impairment (serum creatinine concentration, 4 mg/dL), underwent non-contrast-enhanced CT, which demonstrated rupture but no endoleak. In 1 of 2 patients who did not undergo CT a technical error occurred during the original operation, in which the contralateral limb barely reached the native iliac artery. The resulting type I endoleak was noted on the routine 48-hour postoperative contrast-enhanced CT scan. The aneurysm ruptured on postoperative day 4, before planned secondary repair. The clinical signs were such that no further CT scans were considered necessary. In the remaining patient an endoleak was demonstrated on CT scans after deliberate distal deployment because of abnormal anatomy of the renal artery before rupture, and an aortogram was considered to provide more information than another CT scan.

Table I. Manufacturers of endografts implanted before aneurysm rupture

Graft attachment device (White/Yu)	5
Stentor/Vanguard (Mintec Marseille/Boston Scientific)	4
AneuRx (Medtronic)	6
Talent (Medtronic)	2
Endologix (Endologix, Inc)	1
Total	18

Table II. Configuration of endografts implanted before aneurysm rupture

Bifurcated	14
Aortouniiliac	2
Tubular	2
Total	18

The 8 patients with known endoleaks, including the above 2 patients who did not undergo CT at presentation, had primary type I leaks. The source of the endoleak in the remaining 9 patients was migration in 5 patients and expansion of native common iliac artery in 1 patient plus the 3 patients with previous endotension. CT scans in these 3 patients demonstrated endoleak, which was indeterminate in 1 patient, proximal type I in 1 patient, and due to modular disconnection producing type III endoleak in the remaining patient. In 5 patients with migration ($\geq$1 cm) the body of the endograft remained within the neck of the aneurysm in 3 patients, but was free in the sac of the aneurysm in the remaining 2 patients. AneuRx (n = 4) and Endologix (n = 1) prostheses were implanted in this subset of patients with migration. Retroperitoneal hematomas in group 1 patients were similar in site and size with those in group 2 patients.

Open repair of ruptured AAAs. All patients underwent open repair, with the exception of 4 patients in group 1 and 3 patients in group 2, who underwent endovascular repair of ruptured AAAs. Open repair was performed via a transperitoneal approach with the patient under general anesthesia. Supraceliac clamping was used for initial control in 3 of 14 patients in group 1, and supraceliac clamping or compression was used for initial control in 49 of 88 patients in group 2.

Endovascular repair of ruptured AAAs. The 4 patients in group 1 who underwent endovascular repair had proximal type I endoleak (n = 2), distal type I endoleak in an ectatic common iliac artery (n = 1), and modular type III endoleak (n = 1). These were treated with deployment of a proximal aortic cuff, placement of a secondary aortic endograft, iliac limb extension, and placement of an inter-

Table III. Characteristics of patients in groups 1 and 2

	Group 1 (N = 18)		*Group 2 (N = 91)*	
	n	*%*	*n*	*%*
Mean age (y)	74		73	
Sex (F:M)	0:18		27:64	
Maximum AAA diameter (mean in cm)	6.9		6.2	
Ischemic heart disease	10	55	46	51
Hypertension	6	33	35	38
Diabetes mellitus	2	11	7	8
Renal impairment	4	22	11	12
Chronic obstructive pulmonary disease	1	6	11	12

Table IV. Comorbid conditions in 10 patients in group 1 considered to make them unfit for open repair at primary operation

Severe cardiac disease	
ASA stage 3 or 4	8
Renal failure	
Successful transplantation	2
Serum creatinine >2.3 mg/dL	2
Previous stroke	3
Chronic lymphatic leukemia	1
Severe respiratory disease	
Boushy category III*	1
Hostile abdomen†	1
	18

ASA, American Society of Anesthesiologists.
*Boushy: classification of grade of dyspnea modified.[2]
†Obesity, stoma, and adhesions from previous rupture of sigmoid colon due to carcinoma.

position endograft, respectively (Fig). The Figure illustrates the limitations of surveillance with annual CT scanning. Rupture occurred within 10 days of an annual scan in which no endoleak was detected. The case also illustrates the importance of plain x-ray films in demonstrating separation of radiopaque markers at the site of modular connection.

Endovascular repair of ruptured AAAs was limited to 4 patients in group 1 and 3 patients in group 2 because of logistics. Until 2003 at our institution, emergency vascular procedures were performed in another building, in general surgical operating rooms without adequate radiologic equipment or nursing staff familiar with endovascular interventions.

In the 3 patients in group 2 who underwent endovascular repair bifurcated endografts were deployed.

Outcome. Criteria used to assess outcome included mortality, operative blood loss, and duration of stay in the intensive care unit (ICU).

Statistical analysis. Statistical analysis of proportion was performed with the χ^2 test and the Fisher exact test when expected cells were less than 5. Differences in means were tested with the Mann-Whitney *U*test.

RESULTS

The proportion of patients with hypotension at presentation in group 1 (4 of 18) was significantly less than in group 2 (76 of 91; $P < .01$).

Operative findings. The 4 patients in group 1 with hypotension at presentation underwent open AAA repair. All 4 patients had proximal type I endoleaks and retroperitoneal ruptures. Three of 14 patients in group 1 who underwent open AAA repair had anterior rupture of the aneurysm. Rupture sites had some hemoserous fluid trickling from them, but no bleeding under pressure, despite stable hemodynamic conditions. The remaining 11 patients in group 1 and 88 patients in group 2 who underwent open AAA repair had retroperitoneal hematomas. In the patient with previous endotension in whom CT scans demonstrated an endoleak of indeterminate nature, the leak was found to be a type II lumbar endoleak at open exploration.

Outcome in group 1. One of 3 perioperative deaths occurred in the operating room. This patient was 1 of 2 with the endograft free within the aneurysm sac. The remaining 2 deaths were cardiac-related, and occurred on postoperative days 3 and 14, respectively. All 3 patients had undergone open AAA repair.

Outcome in group 2. Of the 49 perioperative deaths, 8 occurred in the operating room and another 7 occurred within the first 48 postoperative hours. Three patients who were successfully resuscitated after cardiac arrest in the emergency department underwent open repair, but none survived longer than 1 week. In addition to the 49 perioperative (30 day) deaths there was 1 in-hospital death, at 40 days postoperatively.

Comparison of outcome in groups 1 and 2. The difference in perioperative (30 day) mortality rate in group 1 (3 of 18; 16.6%) compared with group 2 (49 of 91; 53.8%) was significant ($P < .01$, Fisher exact test). When excluding the 7 patients who underwent endovascular repair of ruptured AAAs, the difference in perioperative (30 day) mortality rate in group 1 (3 of 14; 21%) compared with group 2 (49 of 88; 56%) was significant ($P < .02$, Fisher exact test). The outcome in group 1 was therefore superior to that in group 2.

Nine of 10 patients considered unfit for open repair at the primary operation survived intervention to treat rupture. Eight of the 10 patients underwent open repair; 7 survived, compared with 2 of 2 who underwent endovascular repair.

Blood loss was less in group 1 compared with group 1 (Table V). This difference was significant when all patients were included in the analysis (2588 mL vs 4056 mL; $P < .01$) and also when 7 patients in whom ruptured aneurysms were treated with endovascular repair were excluded (3214 mL vs 4178 mL; $P < .02$).

The difference in duration of ICU stay between group 1 and group 2 was not significant, irrespective of whether analysis included all patients or excluded those in whom ruptured AAAs had been repaired with the endovascular method.

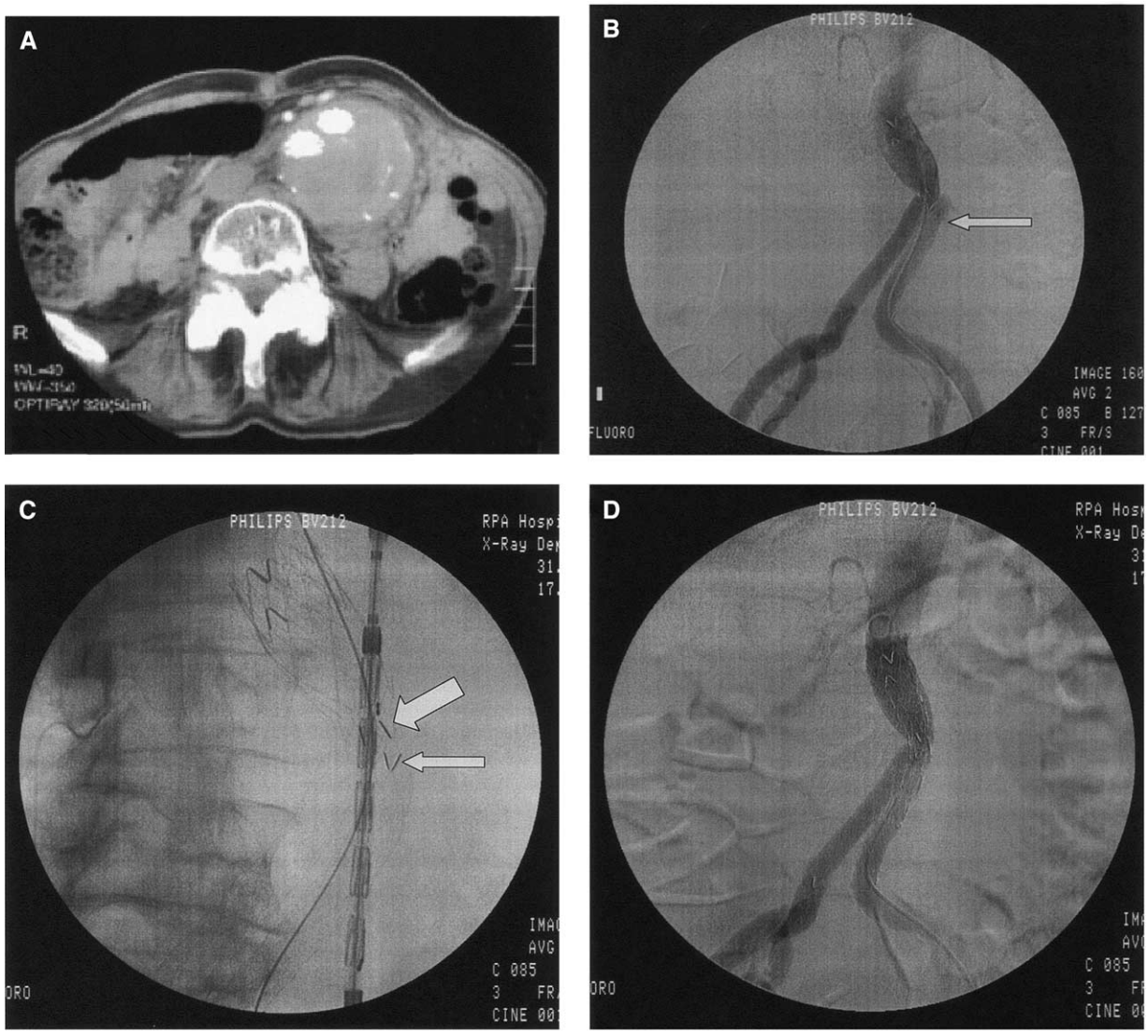

Fig 1. This case illustrates the limitations of surveillance with annual scans and the importance of plain x-ray films in demonstrating separation of radiopaque markers. The patient underwent endovascular abdominal aortic aneurysm (AAA) repair with a Vanguard prosthesis in 1996. Preoperative diameter was 5.4 cm. At follow-up, diameter was 5.00 cm (1998), 5.2 cm (1999), 5.5 cm (2000), 5.5 cm (2002), and 6.1 cm (2003). No endoleak was demonstrated in these studies. **A,** Contrast material–enhanced computed tomography scan of AAA (diameter, 6.5 cm) demonstrates endoleak and rupture, obtained 10 days after annual scan in 2003, which demonstrated no endoleak. **B,** Aortogram demonstrates type III endoleak between contralateral stump and contralateral limb *(arrow)*. **C,** Predeployment plain film for orientation of secondary endograft. Note markers on contralateral stump *(broad arrow)* and contralateral limb of primary endograft *(narrow arrow)* are no longer adjacent, indicating migration. **D,** Postprocedure aortogram confirms exclusion of sac from the circulation.

DISCUSSION

Since the 1950s, when open graft repair of AAAs was instituted, there have been advances that have reduced the mortality rate for elective AAA repair to less than 5%.[3,4] Recent reports of Food and Drug Administration trials have reported mortality rates of 0% for open AAA repair.[5,6] Mortality rates for operative repair of ruptured AAAs, however, have remained consistently high, between 40% and 50% during this period.[4,7,8] A mortality rate of 16.6%, therefore, in treatment of ruptured AAAs after previous endovascular repair is not only superior to the treatment of AAAs that ruptured without previous treatment in the present study, but demonstrates marked improvement compared with these published results. It should be noted, however, that the proportion of patients with rupture in the present study treated with endovascular repair was smaller

Table V. Outcome of AAA rupture in each group

	Group 1 (N = 18)		*Group 2 (N = 91)*		
	n	*%*	*n*	*%*	P
Deaths					
All patients	3/18	17	49/91	54	<.01*
Excluding 7 EVAR†	3/14	21	49/88	56	<.02*
Mean blood loss (mL)					
All patients	2588		4056		<.01‡
Excluding 7 EVAR†	3214		4178		<.02‡
Mean duration of ICU stay (day)					
All patients	4.2		4.9		>.59‡
Excluding 7 EVAR†	5.1		4.8		>.11‡

AAA, Abdominal aortic aneurysm; *ICU*, intensive care unit.
†EVAR, ndovascular repair of ruptured AAA.
*Fisher exact test.
‡Mann-Whitney *U* test.

in group 2 (3 of 91; 3%) than in group 1 (4 of 18; 22%). Inasmuch as all 7 patients with ruptured AAAs treated with the endovascular method survived, this is a source of potential bias. When the 7 patients with AAAs treated with the endovascular method were excluded, analysis revealed that the difference in perioperative mortality rate in group 1 (3 of 14; 21%) compared with group 2 (49 of 88; 56%) remained significant ($P < .02$).

The ability of 7 patients originally considered unfit for elective open repair to survive rupture and conversion to open repair is an indication of how different the rupture process was between groups 1 and 2. This may also reflect the lack of precision in our ability to assess risk.

Bernhard et al[9] reported an operative mortality rate of 50% in 6 patients in whom AAAs ruptured after endovascular repair with Guidant/EVT devices. These ruptures had certain unique features; all occurred in patients with tube endografts, and all were a consequence of a type I endoleak that developed at the distal attachment site. Widening of the distal aortic neck and hook fractures were also each thought to be a factor in 5 of the 6 patients. It seems, under these circumstances, therefore, that the previous tube endografts were unlikely to be in a position to have any beneficial effect at the time of rupture. The authors also reviewed 40 additional cases of rupture after endograft repair with devices from a diverse group of manufacturers. The perioperative mortality rate for the combined Guidant/EVT and published cases was 41%. The authors concluded that the outcome of rupture after endograft repair is similar to that expected for patients without previous endografts.

Reporting a series of 18 patients who required operative intervention because of AAA rupture after endovascular repair is not a record to be proud of. It is important to look at the reasons why AAAs in these 18 patients were allowed to progress to rupture, and determine whether future ruptures may be prevented. Three of 8 patients with aneurysms that ruptured in the presence of a previously known endoleak underwent treatment in the early part of the study, with the misguided belief that type I endoleaks might seal spontaneously if given adequate time. This is now known to be incorrect, and current practice mandates that all type 1 endoleaks be treated promptly. Another 3 patients underwent, collectively, 5 unsuccessful endovascular attempts to seal endoleaks. The remaining 2 patients with endoleaks were anatomically unsuitable for further endovascular repair, and were considered unfit for conversion to open repair. Despite this, 1 of the 2 patients survived conversion to open repair after rupture. Improved technology may enable these latter 2 groups to undergo successful endovascular repair of endoleaks. Endotension in 3 patients emphasizes that management of this condition remains a continuing problem. Originally confined to isolated case reports, endotension has now emerged as a major concern after the reported 57% probability at 4 years of AAA enlargement after endovascular repair with the Excluder device.[10] These 3 patients were part of a group of 18 patients with diagnosed endotension during the study. Nine patients have undergone either endovascular intervention or conversion to open repair. The aneurysm sac diameter at which conversion to open repair is advised is not known, but the mean diameter at which these 3 AAAs ruptured was 7.2 cm (range, 6.5-8.0 cm). Two additional patients were overdue for follow-up. The risk for rupture therefore could, it is hoped, be reduced by awareness of the need to consider treatment of endotension in patients with AAAs exceeding 6.5 cm diameter, and the need for both the attending surgeon and the patient to be scrupulous in ensuring punctuality of follow-up. Careful examination of serial plain x-ray films is an integral component of endograft surveillance in general, but is particularly important in patients with endotension, because it may provide a clue to the site of the problem. Had the patient in the Figure undergone plain radiography in addition to contrast-enhanced CT, which showed no endoleak 10 days before rupture, migration of the contralateral limb marker may have been noted, thus enabling prophylactic intervention. The remaining group of 5 patients who regularly attended follow-up and had no endoleak or expansion of the aneurysm are the most worryisome. The risk for unpredictable rupture is small (99.5% probability of freedom from rupture at 3 years), but remains a cause for concern.[11] Because the goal of endo-

vascular AAA repair, however, is to prevent death from rupture, the findings of the present study may give some comfort to patients who may potentially be included in this group, such that in the event of rupture their chances of surviving secondary AAA repair are reasonably good, at 83%.

It is interesting to speculate on the reasons for this better-than-expected survival in patients with ruptured AAAs after endovascular repair. It seems that the most likely explanation is that these patients are in a relatively stable hemodynamic state at presentation. Only 4 of 18 patients had systolic blood pressure less than 100 mm Hg, compared with 76 of 91 with rupture before any treatment. The findings at presentation and outcome in patients in this study support our earlier hypothesis that seeks to explain these findings.[1] By definition, all patients with rupture after endovascular AAA repair have an endoleak that enables communication between the aortic lumen and the exterior of the aneurysm sac. It is known that endoleak, whether previously known or not, enables systemic arterial pressure to be communicated to the sac of the AAA in the same way as would occur in an untreated AAA. The risk for rupture therefore remains the same in these patients as in those with untreated AAAs. Once rupture occurs, however, the quantity and rate of blood loss outside the aneurysm sac is limited by the endoleak channel. Patients survive AAA rupture because a combination of retroperitoneal pressurization and thrombosis induces temporary hemostasis. These are more likely to be effective if the extravascular channel is long and narrow as a result of previous endovascular AAA repair. In the case of an untreated AAA, however, blood loss is limited only by the defect in the ruptured sac, which has little chance to thrombose. In situations in which the endograft has migrated from the proximal neck of the aneurysm into the sac it is clear that the preceding endovascular repair would have no effect on the outcome of rupture.

An alternative explanation for better-than-expected survival in group 1 could be selection bias. Six patients went to the emergency departments of other hospitals before transfer for surgical repair of their aneurysms. No patients were denied transfer or died in the transfer process, making selection bias unlikely. Patients in group 1, however, were all anatomically suitable for endovascular repair at the primary operation, and would likely have had more favorable anatomy than those in group 2. The incidence of supraceliac clamping for initial control in open repair was greater in group 2 compared with group 1, and this may also be a cause of bias.

The beneficial effect of previous endovascular AAA repair appears to be limited to enabling a stable hemodynamic state at presentation for treatment, and less operative blood loss, compared with patients in group 2. Once open repair was completed, no advantage was observed for patients in group 1 with regard to shorter ICU stay compared with patients in group 2.

The findings of this study confirm that endovascular AAA repair complicated by endoleak does not prevent rupture. The data suggest, however, that rupture, when it does occur in these circumstances, may not be accompanied by such major hemodynamic changes and higher mortality rate as rupture of an untreated AAA. Analysis of a larger group of patients is required to confirm this apparent intermediate level of protection afforded by failed endovascular repair, which does not prevent rupture but enhances survival after operation to treat rupture, possibly by ameliorating the hemodynamic changes associated with the rupture process.

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DISCUSSION

Dr Jon S. Matsumura (Chicago, Ill). I've been fortunate to recently visit the Royal Prince Alfred Hospital and have seen the outstanding work done in this unit. I've even reviewed some of the films and treatment of patients with a rupture, and I have no doubt about the accuracy of this report: 83% of the patients survived rupture.

This contrasts with Dr Bernhard summary of the world literature of rupture, which showed a 59% survival, and your own experience with de novo rupture, 46%. So I agree there is some intermediate protection, but you're doing even better than that. And the easiest explanation is that you're better at treating these patients. But I search for other possible covariates. Specifically, was

there a difference in time from onset of symptoms until definitive treatment? A difference such as might be explained by increased sensitivity of the patients to symptoms in intensive follow-up after EVAR. Or is your group more effective at preemptively re-treating patients who are at risk for the really big endoleaks and the potentially fatal ruptures?

My last question is a bit philosophical. If 83% of your patients survive a rupture after an endovascular graft, and if the mortality with elective conversion is comparable to that, do you now recommend against avoiding open conversions in that scenario?

Dr James May. In answer to your questions, first, the onset of symptoms did appear to be longer in patients who had had a previous endovascular repair, not in all of them, but certainly some of them had symptoms for a couple of days before they even presented to hospitals, so I think that could be a factor.

I don't think retrieval was a factor, because some of them still took quite a while to actually get to us.

And finally, I think your question about the survival rate and these patients surviving conversion is a good one, because 10 of 18 of our patients we thought at the initial operation were unfit for open repair, and despite that, 9 of them survived. So I think it probably says a lot for how inexpert we are at actually assessing the risk in these patients.

Dr Jacob Buth (Eindhoven, The Netherlands). Was there any difference in mortality between the post-EVAR rupture patients with endovascular treatment or with open treatment? In other words, were all endovascular approaches successful, which would automatically increase mortality in the open procedures?

Dr May. Yes, that's quite correct. All 7 patients who were treated with endovascular repair survived, and that's why it was necessary to remove that bias by excluding them in the analysis.

Dr Buth. I think the EUROSTAR experience would confirm these observations, because mortality in the patients who underwent secondary endovascular repair of rupture was significantly better in the whole group.

Dr Anthony J. Comerota (Toledo, Ohio). I was surprised at the results of your ICU stays between the 2 groups. If you excluded on-table deaths and very early operative mortality in your open repair group or the frank rupture group, would that have made a difference in ICU stay, because there's an inherent bias built into that type of analysis.

Dr May. I don't think that it would make a difference. I would not have expected a difference in ICU stay, because once the patient has embarked on open repair and that open repair is completed, I can't see how the previous endovascular repair could have very much influence on the course at that stage.